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QRS notch timing in idiopathic outflow tract premature ventricular complexes

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ABSTRACT

Background and aims: The precise preprocedural localization of outflow tract premature ventricular contractions (OT-PVCs) remain essential yet challenging, owing to the region's complex and variable anatomy, particularly for left-sided origins. Although numerous electrocardiographic localization algorithms have been proposed, most are constrained by a trade-off between diagnostic accuracy and clinical practicality. QRS notching is a frequently observed but largely overlooked feature in OT-PVCs, and it remains understudied in the context of idiopathic ventricular arrhythmias. This study aims to move beyond a simple analysis of notch distribution across leads by leveraging QRS notching as a functional marker of ventricular activation to enhance site of origin localization.
Methods: This retrospective observational study included 105 patients who underwent successful catheter ablation for symptomatic OT-PVCs. The cohort consisted of 57 right ventricular outflow tract (RVOT) and 48 left ventricular outflow tract (LVOT) PVCs. All PVCs exhibited a left bundle branch block morphology with an inferior axis, defined by a dominant R-wave in the inferior leads and a QS complex in leads aVL and aVR. Furthermore, each PVC demonstrated a single, reproducible notch with a duration of $\leq$20 ms. Notch Timing (NT) was defined as the interval from the earliest onset of the global PVC QRS complex to the nadir of the identifiable notch. When notches were asynchronous across the 12 leads, the timing of the latest-occurring notch was recorded for analysis. The diagnostic performance of the NT criterion for differentiating RVOT from LVOT origins was evaluated and compared against the V2S/V3R index using receiver operating characteristic (ROC) curve analysis, with the area under the curve (AUC) serving as the primary metric of comparison.
Results: Of 105 patients (45.6 $\pm$ 13.4 years, 55% male), 57 had RVOT and 48 had LVOT PVCs. NT robustly differentiated origins, with RVOT PVCs demonstrating significantly later notches than LVOT PVCs (94.3 $\pm$ 15.0 ms vs. 74.5 $\pm$ 15.6 ms, $p < 0.001$). An NT >80 ms favored an RVOT origin with 87.7% sensitivity and 68.8% specificity (PPV = 76,9%, NPV = 78,1%, AUC 0.838). The diagnostic performance of NT was comparable to the established V2S/V3R index (AUC 0.828). Characteristic lead distribution patterns augmented localization, though extensive notching across both the inferior and lateral precordial leads was not specific for a free-wall RVOT origin.
Conclusion: This study introduces the first structured framework for QRS notch analysis—defining its characteristics, timing, and distribution—to improve arrhythmia localization. This represents a shift from descriptive morphology to mechanistic interpretation, where a novel notch timing metric robustly discriminates RVOT from LVOT origins by quantifying delayed transeptal conduction.

Introduction

The outflow tracts constitute a critical hub of arrhythmogenesis, particularly for idiopathic premature ventricular contractions (PVCs) [1]. However, precise pre-procedural localization of their site of origin remains a significant challenge. Localization is of clinical importance, as PVCs arising from the left ventricular outflow tract (LVOT) require a more cautious and tailored ablation strategy owing to the proximity of coronary arteries and other critical structures, thereby increasing procedural risk [1,2]. This ongoing clinical challenge continues to drive the

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search for more reliable and discriminative electrocardiographic markers to improve pre-procedural localization [2]. Numerous electrocardiographic (ECG) algorithms have been developed but they present a persistent trade-off between diagnostic accuracy and clinical practicality [2].

The QRS notch is distinctively defined not as a full waveform, but as a brief deflection or interruption in the continuity of the QRS complex [3]. Although QRS notching is a commonly observed phenomenon in PVCs, it is frequently dismissed in clinical practice as an incidental finding or simply absorbed into broader morphological descriptions [4–6]. While prior studies have associated specific notch patterns—such as notched inferior leads with RVOT free-wall origins [5,6] or a V1 downslope notch with aortic cusps junction [4]—with PVC localization, a systematic analysis of the notch itself is absent. This study addresses this gap by rigorously characterizing QRS notching in OT-PVCs, transforming it from a descriptive feature into a practical localization tool through the novel integration of notch timing and lead distribution.

Methods

Study protocol

This retrospective observational study evaluated a cohort of patients who underwent catheter ablation for symptomatic idiopathic OT-PVCs between 2023 and 2025. Inclusion required a PVC morphology consistent with an outflow tract origin defined by a dominant, exclusive R-wave in the inferior leads (II, III, aVF) and a QS complex in leads aVL and aVR. Furthermore, the presence of a single, reproducible QRS notch ($\leq$ 20 msec) was required in every PVC complex. Exclusion criteria comprised non-OT PVC origins, polymorphic ectopy (morphology or axis variation in at least 2 leads), the absence of a notch, fragmented QRS (multiple notches within the same lead), and notch durations >20 msec (considered a distinct wave). Of the initial cohort, 105 patients met all the criteria.

ECG analysis

Standard 12-lead ECGs were acquired during the PVC ablation procedure using an electrophysiology recording system (Prucka CardioLab, GE Healthcare). All ECG measurements were performed using digital calipers at a sweep speed of 50 mm/s. Variables measured included the QRS duration, which was determined using the global QRS method to identify the earliest onset of the QRS complex across all 12 leads; this served as the reference point for notch timing measurements. The Notch timing (NT) was defined as the interval from the beginning of the QRS complex to the nadir of the identifiable notch across all 12 leads (Fig. 1). If the notch was not synchronous across the leads, the timing of the latest notch was recorded. The difference in notch timing between leads was assessed when notches were asynchronous, and the specific leads in which the notch was identified were documented.

A Notch Score was calculated to quantify the spatial distribution of notching across anatomically grouped leads. The score was derived by assessing four distinct lead groups: the inferior leads (II, III, aVF), leads aVL and aVR, the early precordial leads (V1–V2), and the lateral precordial leads (V3–V6). Each group was assigned 1 point if one or more leads within it exhibited a qualifying notch ($\leq$20 ms), and 0 points if no notch was present. The total Notch Score (range 0–4) thus represented the number of lead groups involved.

Additional ECG variables were analyzed: The peak-to-peak amplitude and qualitative morphology in Lead I, the ratio of QS-wave amplitudes in leads aVL and aVR (AVL/AVR ratio), the ratio of R-wave amplitudes in leads II and III (II/III ratio), and the V2S/V3R index. Qualitative assessment of V1 morphology (e.g., RS, QS, R, Rs, Qr) was performed. The precordial transition was defined as the first precordial lead where the R-wave amplitude exceeded the S-wave amplitude. The difference in transition zone between the PVC and sinus rhythm was scored as 0 if in the same lead, negative if earlier in the PVC, or positive if later.

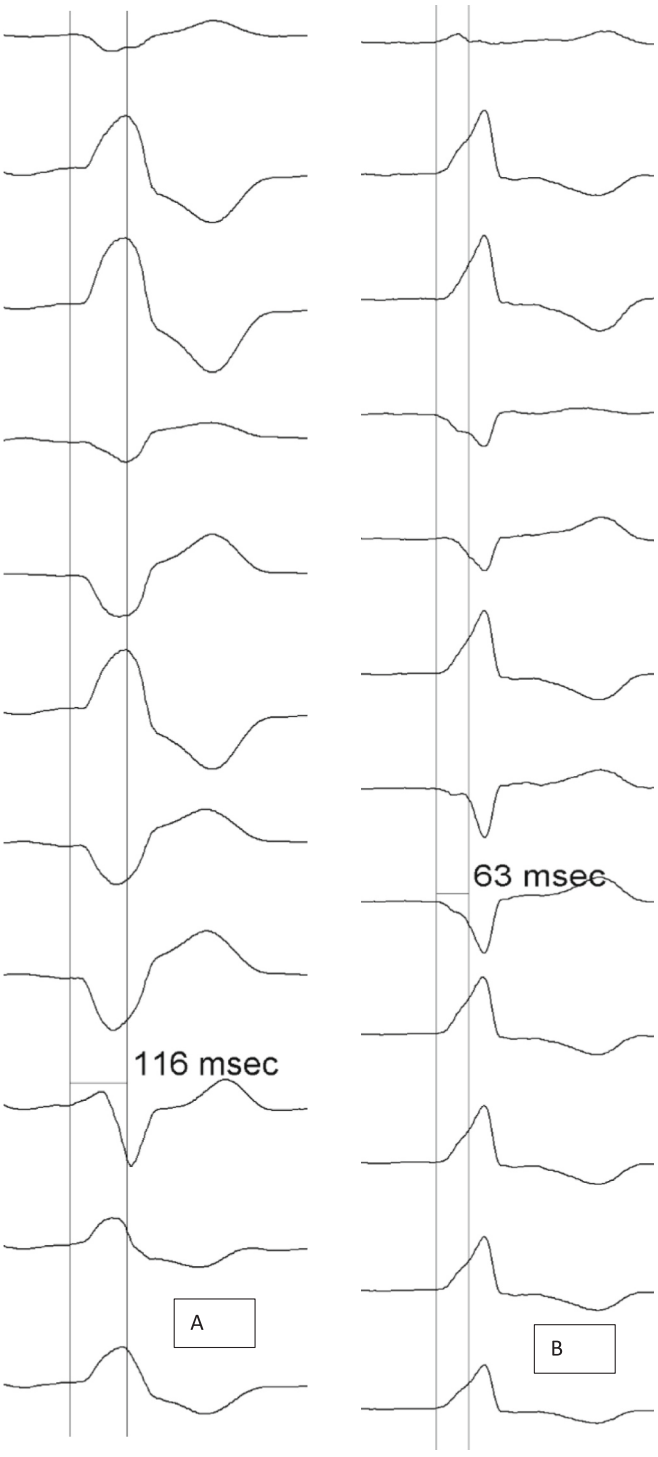

Figure 1: Notch Timing measurements
A. RVOT PVC
B. LVOT PVC

Fig. 1. Notch timing measurements A. RVOT PVC B. LVOT PVC.

Electrophysiological and electroanatomical analysis

All procedures were performed using a 3D electro-anatomical

mapping (EAM) system (CARTO 3, Version 7.5, Biosens Webster). Mapping and ablation were conducted using a 3.5 mm tip irrigated radiofrequency ablation catheter (STSF). A decapolar catheter was positioned within the coronary sinus and advanced into the great cardiac vein in all patients. Activation mapping was performed during PVCs, with the earliest ventricular activation site identified based on the earliest bipolar electrogram. Manual annotation of the earliest bipolar and unipolar electrograms was performed in all cases.

The initial mapping strategy focused on the right ventricular outflow tract (RVOT) when the local ventricular activation preceded the surface PVC QRS by a significant interval (5–10 ms). If a broad zone of early activation was encountered, comprehensive mapping was extended to the left ventricular outflow tract (LVOT), including the aortic valve cusps. Radiofrequency ablation was targeted at the site of earliest bipolar activation. When the earliest activation was recorded in the distal coronary sinus/great cardiac vein, suggesting a left ventricular summit origin, and endocardial radiofrequency ablation failed to eliminate the PVC, ethanol infusion was pursued. This involved obtaining a coronary venogram to identify a suitable cardiac vein for targeted mapping (guide wire mapping)and subsequent alcohol delivery [7].

Coronary angiography was performed during LVOT mapping in all cases. The successful ablation site—defined as the point of earliest ventricular activation where radiofrequency application resulted in complete PVC elimination—was taken as the definitive SOO. Sites were classified as right-sided OT (RVOT), including septal or free-wall locations; left-sided OT (LVOT), including the coronary cusps, left ventricular summit, and aorto-mitral continuity; or intramural PVCs (IM-PVC). Intramural PVCs were defined anatomically as anterior (Free wall), mid, or septal intramural [8], with mapping criteria including equally early bipolar electrograms on both sides of the septum and ablation failure despite optimal contact and impedance drop.

Statistical analysis

All ECG measurements and EAM data were entered into a dedicated database and analyzed using SPSS software. Descriptive statistics were used to summarize the data; continuous variables are expressed as mean ± standard deviation or median [interquartile range], while categorical variables are presented as frequencies and percentages. The primary analysis involved constructing a Receiver Operating Characteristic (ROC) curve to evaluate the ability of notch timing (NT) to differentiate between RVOT and LVOT origins. The optimal cut-off value was determined using Youden's index, and sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated. A comparative analysis was performed to assess the diagnostic performance of the NT cut-off against the V2S/V3R index, by comparing the areas under the ROC curves (AUC). Subgroup analyses were conducted within the RVOT and LVOT groups to compare NT values across specific anatomical subgroups using appropriate tests. Associations between NT and other ECG morphological characteristics were analyzed using chi-square or correlation tests. A two-tailed *p*-value of less than 0.05 was considered statistically significant for all analysis.

Results

Patient characteristics

The study cohort comprised 105 patients with OT-PVCs. The mean age was 45.6 ± 13.4 years, 55% were male, and the mean left ventricular ejection fraction (LVEF) was 57.9% ± 7.7%. Comorbidities had a low prevalence: four patients had ischemic heart disease (two of whom had intramural LVOT PVCs) and four had diabetes mellitus.

48 PVCs originated from the LVOT (distributed in left coronary cusp [LCC] = 12, right coronary cusp [RCC] = 2, right–left cusp junction [R-LCC] = 5, aorto-mitral continuity [AMC] = 1, left ventricular summit [LVS] = 13, intramural LVOT [IMLVOT] = 15) while 57 originated from the right ventricular outflow tract (RVOT) (distributed in: anterior free-wall RVOT [AFWRVOT] = 12, right free-wall RVOT [RFWRVOT] = 4, left free-wall RVOT [LFWRVOT] = 17, mid-septal RVOT [MSRVOT] = 8, left septal RVOT [LSRVOT] = 15, pulmonary cusp [PC] = 1).

Patients with LVOT focus were significantly older (mean ≈50.1 ± 12 years vs 41.5 ± 13 years for RVOT, $p = 0.003$) and had a lower LVEF compared to RVOT origin (55.7% ± 10 vs 59.9% ± 4.0 for RVOT, $p = 0.013$); all ischemic cardiomyopathy cases were in the LVOT group (but LVEF was preserved in all of them) (Table 1).

Electrocardiographic analysis

There was no significant difference between RVOT and LVOT PVCs' QRS duration (142.9 msec vs 144.4 msec; $p = 0.60$). Amplitude ratios aVL/aVR (1.21 ± 0.55 vs 1.08 ± 0.35; $p = 0.16$) and II/III (0.96 ± 0.17 vs 0.97 ± 0.18; $p = 0.70$) did not differ significantly between groups, suggesting many PVCs occupy closely related outflow regions (Table 1).

Precordial transition level was usually the same as sinus rhythm ($n = 58$); earlier transitions tended to indicate LVOT origins and later transitions RVOT origins, with notable exceptions: some septal RVOT sites showed earlier transition ($n = 9$) and some LVS/IM LVOT sites produced later transitions ($n = 3$). Morphologically, most features were similar between groups, but an isoelectric wave at the onset of V1–V2 was specific for LVOT PVCs, and a qR pattern in lead I was specific for free-wall RVOT, particularly anterior free-wall RVOT.

Notch analysis and localization patterns

Notching was most frequently seen in V3–V6, followed by the limb leads aVL and aVR, lead I, the inferior leads, and then V1 and V2. Notches were predominantly observed at the peak of the R or S wave, with a lower incidence along the ascending or descending segments of those waves. Most PVCs exhibited synchronous notch timing across all ECG leads. When asynchrony was present, the observed inter-lead timing differences ranged from 15 to 25 ms, with Lead I most frequently displaying the latest notch, a pattern attributable to variable cardiac rotation. To standardize measurement against this anatomical variation, Notch Timing was defined by the latest-occurring notch across the 12-lead ECG. This methodological choice was further supported by the consistent temporal alignment of this latest notch with a change in QRS deflection polarity (e.g., S-to-R transition) in the precordial leads. This

Table 1
Patient characteristics analyzed comparatively between the two groups.

Variable	LVOT ($n = 48$)	RVOT ($n = 57$)	*P* value
Age (years)	50.05 ± 12	41.54 ± 13	0.003
Male (%)	50%	45.6%	0.713
LVEF (%)	55.66 ± 10	59.91 ± 4.03	0.013
DM (n)	2	2	0.57
ICM (n)	4	0	0.027
QRS duration (ms)	142,9 ± 15.16	144.39 ± 13.78	0.6
Mean Lead I Amplitude (mv)	0.42 ± 0.18	0.37 ±0.13	0.13
Lead AVL/AVR ratio	1.21 ± 0.55	1.08 ± 0.35	0.16
Lead II/III ratio	0.96 ± 0.17	0.97 ± 0.18	0.7
Transition Sinus level -0 (n)	23	35	0.045
Transition Sinus level -1 (n)	18	9	–
Transition Sinus level +1 (n)	3	10	–
Lead I Notch (n)	30	37	0.8
Inferior leads Notch (n)*	22	26	0.93
aVL/aVR Notch (n)*	38	38	0.26
V1/V2 Notch (n)*	22	15	0.102
V3-V6 Notch (n)*	32	46	0.07
Mean SV2/RV3 index	0.92 ± 0.47	1.78 ± 0.79	<0.001
Mean Notch Timing (NT)	74.5 ± 15.6	94.33 ± 15	< 0.001

LVEF: Left ventricular ejection fraction, DM: Diabetes mellitus, ICM: ischemic cardiomyopathy, ms = milliseconds, mv = millivolts, NT = Notch Timing. LVOT = Left ventricle outflow tract. RVOT = Right ventricle outflow tract, n = number of patients, * = At least one lead with notch.

concurrence suggests the selected notch marks a key inflection point in ventricular activation, reinforcing its physiological relevance as a potential surface ECG correlate of transseptal conduction and a shift in wavefront orientation.

Notch in lead I was particularly common in left-septal and adjacent sites (LSRVOT: 13/15; IMLVOT: 9/15, MSRVOT (7/8), LVS (9/13) and LCC (8/12)). High multi-lead inferior notching (all the 3 inferior leads) was especially associated with AFWRVOT (10/12) and intramural LVOT (9/15). The presence of aVL and/or aVR notching—especially when accompanied by lead I notching—favored septal or left-adjacent origins (LSRVOT, LCC, FWRVOT). (Table 2) Marked multi-lead V3–V6 notching (3–4 contiguous leads) was characteristic of AFWRVOT and IMLVOT; widespread contiguous lateral precordial notching therefore suggests either an anterior free-wall RVOT projecting laterally or an intramural LVOT/summit focus with broad epicardial spread (see Table 2, and supplementary materials Table 1).

In practical terms, the combination patterns are informative: lead I ± aVL/aVR notching but few inferior-lead notches suggest LSRVOT/LCC or nearby left-septal sites; multiple inferior-lead notches plus multi-lead V3–V6 notching strongly suggests AFWRVOT or intramural LVOT/LVS; and extensive four-lead V3–V6 notching favors IMLVOT or AFWRVOT—intramural LVOT in this cohort frequently demonstrated both lead-I involvement and broad V3–V6 notching.

The Notch Timing (NT) analysis

The Notch Timing (NT) separated LVOT from RVOT origins: mean NT was 74.5 ± 15.6 msec in LVOT versus 94.3 ± 15.0 msec in RVOT ($p < 0.001$). The NT demonstrated strong diagnostic performance (AUC = 0.838, overall modal quality ≈ 0.76), which improved when epicardial PVCs (Pseudo-delta >33 msec) were excluded (AUC = 0.863, modal quality ≈ 0.79) (Fig. 2), reflecting the influence of slow pseudo-delta waves in epicardial foci.

Using an NT cut-off of 80 msec favored RVOT when >80 msec, yielding sensitivity 87.7%, specificity 68.8%, positive predictive value 76.9%, negative predictive value 82.5% and a Youden index of 0.533. Among anatomic subgroups, free-wall RVOT showed the highest mean NT (~103 msec), followed by septal RVOT (~93 msec), then epicardial and RCC origins, with LCC and intramural sites displaying lowest NT values (Figs. 3, 4).

In the present study, we identified a revised discriminative cutoff of 1 msec for the SV2/RV3 index, which yielded a sensitivity of 82.2% and a specificity of 71% in distinguishing LVOT from RVOT sites, differing from the value reported in the original study [9].

Intramural PVC characteristics

Intramural PVCs merit separate mention: the intramural group (n = 15 IMLVOT, 14.3% of the cohort) had an older median age (~54 years) and lower LVEF (~48%) than the overall cohort, mean QRS ≈ 143 msec, and were reported to have mean NT value was 68 ± 10 msec (Minimum = 59 msec, Maximum = 95 msec). Precordial transition most occurred at lead V3 (70% of cases), and in half of the patients (7/15), the transition coincided with that observed during sinus rhythm.

Within intramural cases, four were mid-intramural and 11 were free-wall intramural; two of the four ischemic patients had intramural PVCs. The Notch distribution was particularly high in leads AVL and AVR, in septo-lateral precordial leads (V3-V6), and in the inferior leads. (Table 2, and supplementary materials Table 2).

Table 2
Notch location characteristics analyzed comparatively between the three groups.

Notch Location	Right sided n (%)	Left sided n (%)	Intramural n (%)	p value
Lead I	37 (64.9%)	21 (63.6%)	9 (60.0%)	0.98
Inferior	26 (45.6%)	12 (36.4%)	10 (66.7%)	0.11
aVL–aVR	37 (64.9%)	25 (75.8%)	14 (93.3%)	0.07
V1–V2	14 (24.6%)	16 (48.5%)	7 (46.7%)	0.21
V3–V6	45 (78.9%)	20 (60.6%)	13 (86.7%)	0.18

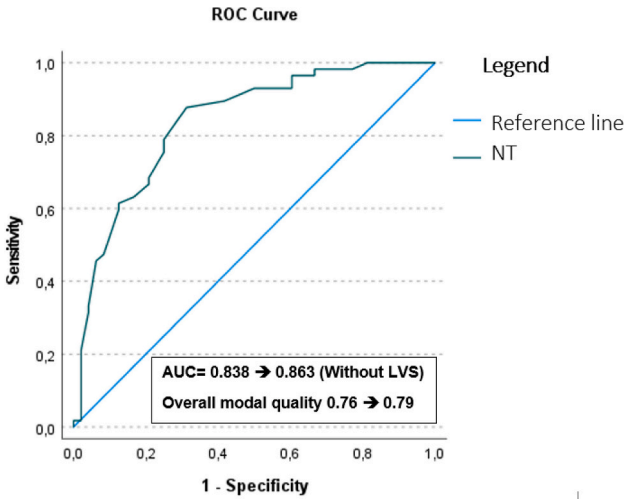

Fig. 2. Notch Timing (NT)' s ROC curve.

Discussion

To our knowledge, this is the first study to systematically analyze and leverage QRS notching in idiopathic OT-PVCs. Unlike prior studies [4–6,10–14], which has largely limited notch assessment to descriptive or topographic lead distribution, we sought to move beyond morphology alone. First, we proposed a pragmatic and reproducible definition of the QRS notch suitable for routine surface ECG analysis at standard recording speeds (50 mm/s). We then performed a comprehensive evaluation of notch characteristics, including its lead distribution, spatial location, and temporal behavior within the QRS complex. Second, in analogy to Treger JS et al.'s notch time in left bundle branch block [15], we introduced notch timing (NT), defined as the interval from QRS onset—determined using the global QRS method—to the nadir of a brief (<20 ms) and unique notch. We assessed the diagnostic performance of NT for discriminating among different PVC sites of origin using ROC analysis, AUC, and the Youden index, and compared its performance with established ECG metrics.

In this cohort of 105 patients with idiopathic outflow-tract PVCs, notch timing (NT) emerged as a robust discriminator of PVC site of origin. NT was significantly longer in RVOT compared with LVOT PVCs (94.3 ± 15.0 ms vs. 74.5 ± 15.6 ms; $p < 0.001$), despite similar global QRS duration and no significant differences in the II/III and aVL/aVR amplitude ratios between groups, indicating that these PVCs originated from closely related and overlapping outflow-tract regions. NT demonstrated strong diagnostic performance for RVOT localization (AUC = 0.838), which further improved after exclusion of epicardial PVCs (AUC = 0.863), underscoring the influence of pseudo-delta conduction on early QRS components. A NT cut-off of 80 ms favored an RVOT origin with high sensitivity (87.7%) and acceptable specificity (68.8%), performing comparably to the established V2S/V3R index, including in the challenging subgroup with V3 precordial transition.

The V2S/V3R index was selected as the reference comparator due to its established validation and clinical practicality for OT-PVC localization. While the original study [9] proposed a cut-off of 1.5 mv, our cohort identified an optimal threshold of 1.0 mv, a difference attributable to population-specific anatomical and electrocardiographic factors as well as the clustering of PVC origins within closely related outflow-tract regions in our population. Even with this adjusted cut-off, the index maintained strong diagnostic performance, achieving accuracy

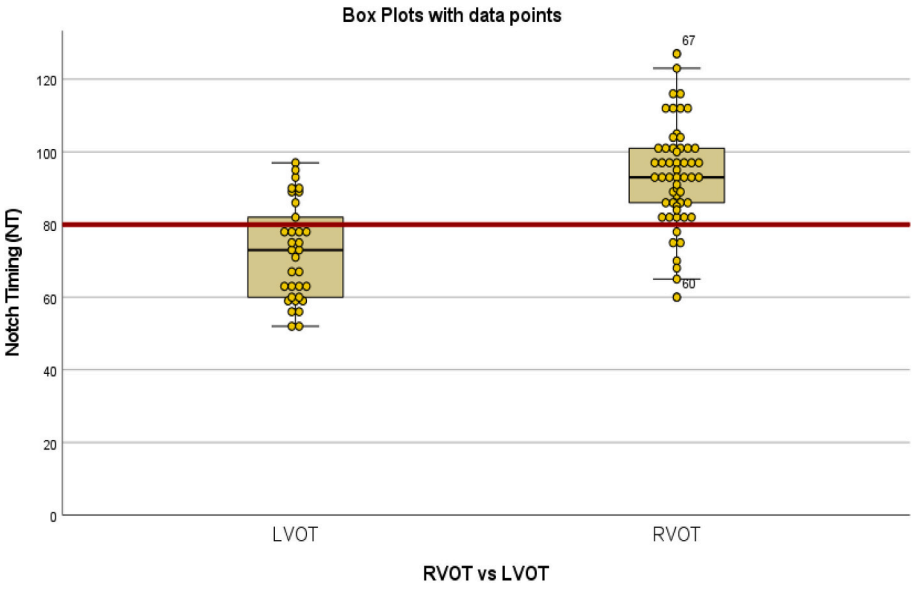

Fig. 3. Notch Timing (NT)' s Box Plot with data points relative to Right sided (RVOT) and to left sided (LVOT).

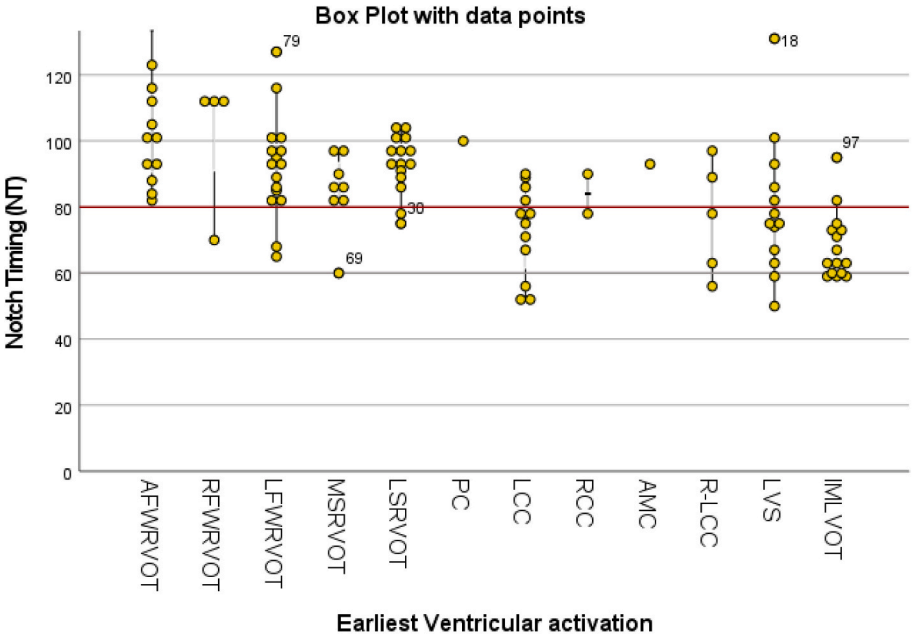

Fig. 4. Notch Timing (NT)' s Box Plot with data points relative to each site of right and left outflow tracts (see text for abbreviations).

comparable to notch timing, especially in cases with a V3 precordial transition.

Subgroup analysis revealed a graded increase in NT across anatomical sites, with the longest values in free-wall RVOT, intermediate values in septal RVOT and coronary cusp origins, and the shortest NT in LVOT and intramural PVCs (Fig. 4). Beyond timing, specific notch distribution patterns offered complementary localization insights (Table 2): Notching in lead I was common across all groups, indicating limited specificity. However, prominent notching in leads aVL and aVR was most frequent in intramural PVCs (93%). A pattern of extensive notching across the lateral precordial leads (V3–V6) was characteristic of both right-sided (79%) and intramural (87%) origins, occurring less often in left-sided foci (61%). These distribution trends, while not statistically definitive in this cohort, suggest that spatial notch analysis can improve PVC focus localization.

QRS notching

Early animal and human studies defined the QRS notch as a discrete change in the slope and direction of an inscribed deflection in the QRS complex which may be unique or multiple [3,16]. These high frequency discontinuities within the QRS complex particularly when multiple have been considered as a marker of structural heart disease (SHD), and were correlated with the presence of anatomical islands of viable myocardial cells in an area of infarct [17].

Early work on QRS notching did not specifically focus on QRS width; however, the presence and significance of notching within wide QRS complexes remain relatively understudied. Available data suggest that when examined, the presence of two or more discrete notches within a wide QRS complex is strongly correlated with myocardial scar [18]. In structurally normal hearts, QRS notching has been most extensively described in left bundle branch block (LBBB) [15,19–21], whereas its

characterization in premature ventricular contractions (PVCs) remains limited. Notably, notches observed in idiopathic outflow-tract (OT) PVCs are typically narrow (<40 ms), distinguishing them from the broader and more fragmented patterns often associated with structural heart disease [18].

Mechanistic insights derived from LBBB offer a useful conceptual framework. In LBBB, QRS notching is attributed to transeptal conduction resulting from sequential rather than parallel depolarization of the right and left ventricles [20]. In this setting, the initial deflection of the notch is thought to represent the transeptal activation wavefront, while the terminal portion of the notch may correspond to completion of transmural depolarization. This characteristic electrocardiographic pattern has gained clinical relevance, being incorporated into refined LBBB definitions and used to predict and guide response to cardiac resynchronization therapy [15,20] as well as left bundle branch area pacing [21].

Building on these observations, we hypothesized that a single, brief notch in idiopathic outflow-tract PVCs reflects transeptal conduction and may therefore serve as a practical electrocardiographic marker to improve non-invasive localization of PVC site of origin and optimize procedural planning for catheter ablation.

This hypothesis directly underpins the inclusion and exclusion criteria of the present study, which were designed to isolate this specific notch phenotype and minimize confounding by structural heart disease or complex conduction abnormalities.

Unlike previous observations, notch distribution was not predictive of site-of-origin localization [5,6], particularly with respect to inferior lead notching, which was observed not only in free-wall RVOT PVCs but also across multiple LVOT sites.

Notably, the notch pattern observed in this study frequently coincided with a concomitant change in QRS deflection in other surface ECG leads (Fig. 5), supporting the hypothesis that QRS notching in idiopathic OT-PVCs reflects transseptal conduction and sequential ventricular activation. This behavior closely parallels the established LBBB paradigm, in which QRS notching represents delayed septal engagement and is exploited to refine the diagnosis of true LBBB. By analogy, we leveraged notch timing (NT) as a quantitative metric to predict the site of origin (SOO) of OT-PVCs in the present study.

Notch Timing

Using this approach, an NT >80 ms was a significant predictor of an RVOT origin, yielding a sensitivity of 87.7% and a specificity of 68.8%. Diagnostic performance further improved after exclusion of epicardial PVCs. NT was systematically and reproducibly measured from the onset of the QRS complex to the nadir of the notch, ensuring consistency across ECG analyses. The observed difference in NT between LVOT- and RVOT-origin PVCs can be explained by distinct anatomical and histological characteristics of the outflow tracts.

Anatomically, the LVOT lies in closer proximity to the interventricular septum than the RVOT, allowing the depolarization wavefront to engage septal myocardium earlier and more efficiently during LVOT activation. In contrast, RVOT activation originates from a more superior and relatively septum-detached position, necessitating longer transeptal propagation before engaging the septum [22].

Histologically, ventricular activation is most efficient when electrical propagation occurs parallel to myocardial fiber orientation [23,24]. The RVOT endocardium and adjacent RV inflow myocardium exhibit predominantly longitudinal myocyte alignment from base to apex across more than 80% of the wall thickness [25–28], whereas the LV

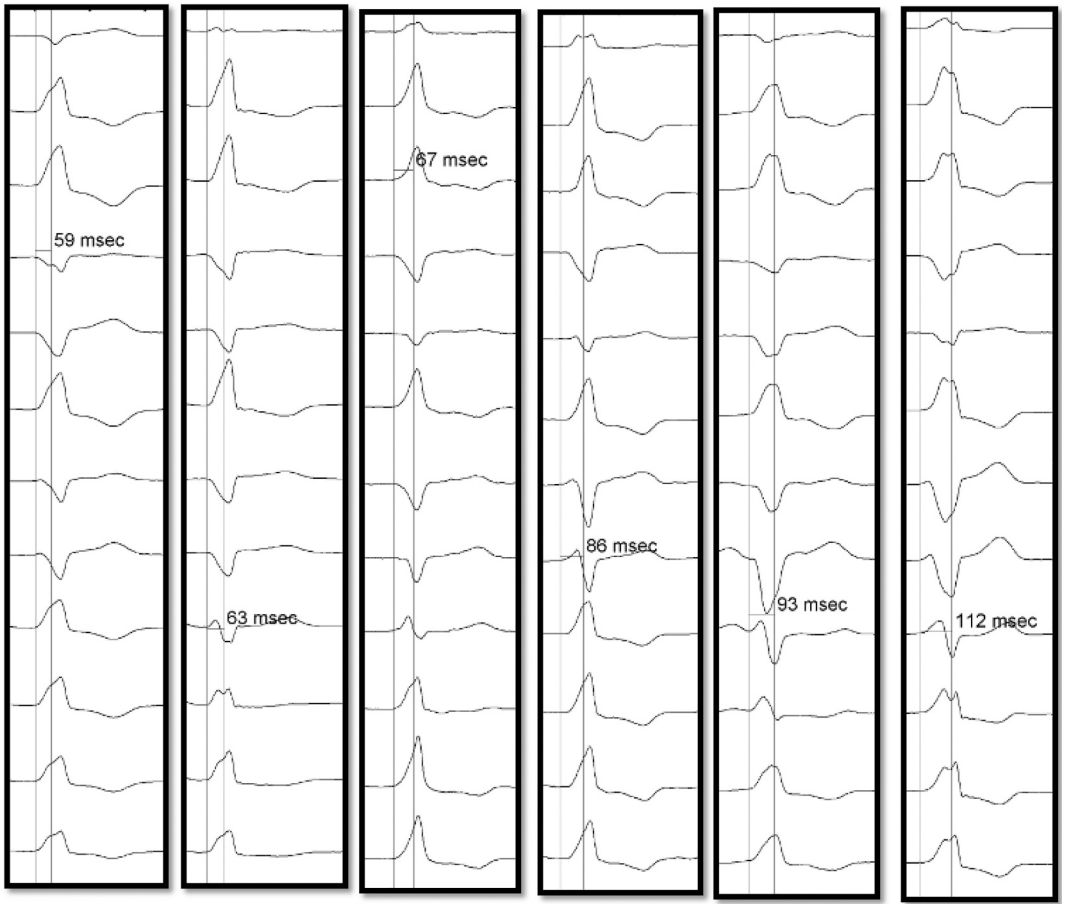

Fig. 5. NT in different outflow tract sites, from left to right: Mid Intramural LVOT, R-L cusps junction, LCC, septal RVOT, Free wall RVOT.

myocardium displays a more complex, multilayered architecture, with obliquely oriented fibers in the sub-endocardium and circumferential fibers in the midmyocardium [26,29,30]. Epicardial fiber organization, however, is relatively homogeneous between the LVOT and RVOT [29]. Functionally, experimental cardiac modulation and mapping studies have shown that RVOT demonstrates prolonged activation times, attributable to slower conduction velocities—potentially in areas of low voltage, with wavefront propagation occurring more preferentially along a longitudinal axis [26,27]. This differs from sites related to the Sinus of Valsalva, which exhibit a more isochronal (simultaneous) propagation pattern [26].

Intramural PVCs

Accurately predicting an intramural (IM) origin from the surface ECG remains challenging, as no single pathognomonic sign exists. Diagnosis therefore relies on synthesizing clinical, electrocardiographic, and invasive mapping data. Accepted criteria for an IM substrate include: equally early bipolar electrograms on both sides of the suspected site (with $\leq \sim$20 msec difference); an initial far-field component at the onset of the bipolar electrogram with a timing mismatch between unipolar and bipolar signals; and the failure of radiofrequency energy to eliminate the focus despite adequate contact and impedance drop-features commonly accepted as indicative of an intramural substrate [8,31,32]. The inherent difficulty in defining IM origins may relate to preferential conduction through insulated fibers within the myocardium [32].

For select free-wall IM cases, targeted mapping within septal perforator vein with alcohol ablation technique, has enabled successful localization and elimination of the PVCs [7].

Although the application of notch timing for intramural premature ventricular contractions did not reach statistical significance, in our experience, intramural foci—particularly those located at mid-septal sites—were associated with shorter notch timing and relatively shorter QRS duration compared to other outflow tract origins. Free-wall intramural PVCs more frequently exhibited a pseudo-delta wave, which altered notch timing and caused subtle changes in lead I morphology (shifting between QS and rS waveforms). Therefore, the combination of a relatively short QRS duration (<140 ms), short notch timing (<70 ms), and a pseudo-delta waveform should raise suspicion for an intramural origin and prompt consideration of dedicated mapping strategies.

NT strengths and limitations

The Notch Timing (NT) approach offers several practical and mechanistic strengths. Because NT is referenced to the global QRS onset and — when notches are asynchronous — uses the latest lead timing, it is inherently robust to variable limb and precordial electrode positioning and to small beat-to-beat shifts in morphology, reducing a common source of surface-ECG error. Measured with digital caliper at 50 mm/s, NT provides a simple, quantitative metric that integrates easily into routine EP lab practice and digital electrogram systems.

In our cohort NT demonstrated strong discrimination between RVOT and LVOT origins (AUC $\approx$ 0.84; improvement to AUC $\approx$ 0.86 after excluding epicardial/pseudo-delta cases) and performed comparably to a well-validated comparator (V2S/V3R), including in the challenging V3 transition subgroup. Beyond binary localization, NT combined with lead-specific notch distribution supplied mechanistic and localization insight, suggesting utility not only as a triage tool for initial mapping but also as a window into substrate geometry.

Several important limitations temper immediate generalizability. The study is single-center and retrospective, with inclusion restricted to patients who underwent successful, mappable ablation and who exhibited a single reproducible notch; this selection strategy introduces potential selection bias and excludes polymorphic or multi-notch PVCs not rarely encountered in practice. Our operational notch definition (unique, <20 msec) and the manual NT measurement method (digital calipers at 50 mm/s) raise reproducibility concerns: borderline, low-amplitude, or multi-component notches may be difficult to classify, and formal inter-observer reliability testing is lacking. Clinically relevant confounders were identified — notably pseudo-delta/epicardial activation increases NT and materially affects diagnostic performance (AUC improved when such cases were excluded) — indicating NT is less discriminative for epicardial substrates and must be interpreted in that context.

Finally, intramural and epicardial substrates are heterogeneous and can blur ECG-anatomy correlations; prospective, multicenter validation (including automated or blinded NT measurement, broader inclusion criteria, and outcome measures such as mapping time, fluoroscopy, and ablation success) will be required to confirm clinical utility and establish reproducibility across recording systems and operators.

Conclusion

This study introduces the first structured framework for the analysis of QRS notches in idiopathic outflow-tract premature ventricular contractions (OT-PVCs), defining their characteristics, timing, and distribution to enhance arrhythmia localization.

Central to this framework is a novel notch timing (NT) metric that robustly discriminates RVOT from LVOT origins by quantifying delayed transeptal conduction, thereby serving as an adjunctive metric for localizing the site of origin of idiopathic PVCs. Further research is needed to prospectively validate NT and broaden its application to other PVC localization.

Clinical trial registration

Not applicable.

CRediT authorship contribution statement

Abdelaziz Jaouadi: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Conceptualization. **Oguzhan Ekrem Turan:** Writing – review & editing, Supervision, Investigation, Formal analysis. **Resit Yigit Yilancioglu:** Supervision. **Umut Inevi:** Software. **Sercan Karaoglan:** Project administration. **Emin Evren Ozcan:** Resources, Investigation, Supervision.

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None.

Declaration of competing interest

The authors declare no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jelectrocard.2026.154361.

Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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